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LETTERS TO THE EDITOR

Anemia and Energy Depletion

The article by Mozaffarian et al. (1) exposes an important principle in cardiovascular physiology that is often overlooked. A prerequisite for efficient energy transfer in living organisms is the interaction of a series of moiety-conserved cycles (2). Cycles improve efficiency and offer an evolutionary advantage (3). Examples of moiety-conserved cycles are the circulation, the Krebs cycle in the mitochondria, and the cross-bridge cycling in the sarcomeres. Anemia deprives the circulation of its most important moiety—red blood cells. From a metabolic perspective, we would like to propose that anemia deprives the body of its primary source of energy when the heart fails to compensate for the decreased amount of red blood cells. Anemia may also contribute to pathologic remodeling of the ventricle in heart failure. Experimental models of anemia have been shown to induce a pattern of metabolic gene expression observed in patients with heart failure (4). This contribution to the progression of heart failure by anemia further impairs the heart's ability to compensate for the decline in red blood cells.

We offer these explanations for the findings of the study by Mozaffarian et al. (1) and also for the recently reported beneficial effects of blood transfusion in elderly patients with acute myocardial infarctions (5).

Heinrich Taegtmeyer, MD, DPhil, FACC
Division of Cardiology
University of Texas
Houston Medical School
6431 Fannin
MSB 1.246
Houston, TX 77030
E-mail: Heinrich.Taegtmeyer@uth.tmc.edu

Saumya Sharma, MD

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REPLY

Drs. Taegtmeyer and Sharma reiterate an important plausible biologic mechanism for the observed association between anemia and higher mortality in severe heart failure. As we also hypothesized (1), diminished oxygen-carrying capacity of anemic blood may necessitate higher cardiac output to meet systemic metabolic requirements; the concomitant increased neurohormonal activation may cause heart failure progression, producing a progressive cycle of metabolic–hemodynamic compromise. This hypothesis is supported by the inverse association between anemia and death due to progressive heart failure, rather than due to sudden death or other deaths (1). However, there are other potential explanations for the observed association between anemia and mortality in heart failure (1). We look forward to testing of this plausible metabolic–hemodynamic hypothesis in ongoing experimental and clinical trials.

Dariush Mozaffarian, MD, MPH
Harvard School of Public Health
665 Huntington Avenue
Building 2, Room 315
Boston, MA 02115
E-mail: darymd@hotmail.com

Wayne Levy, MD, FACC

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